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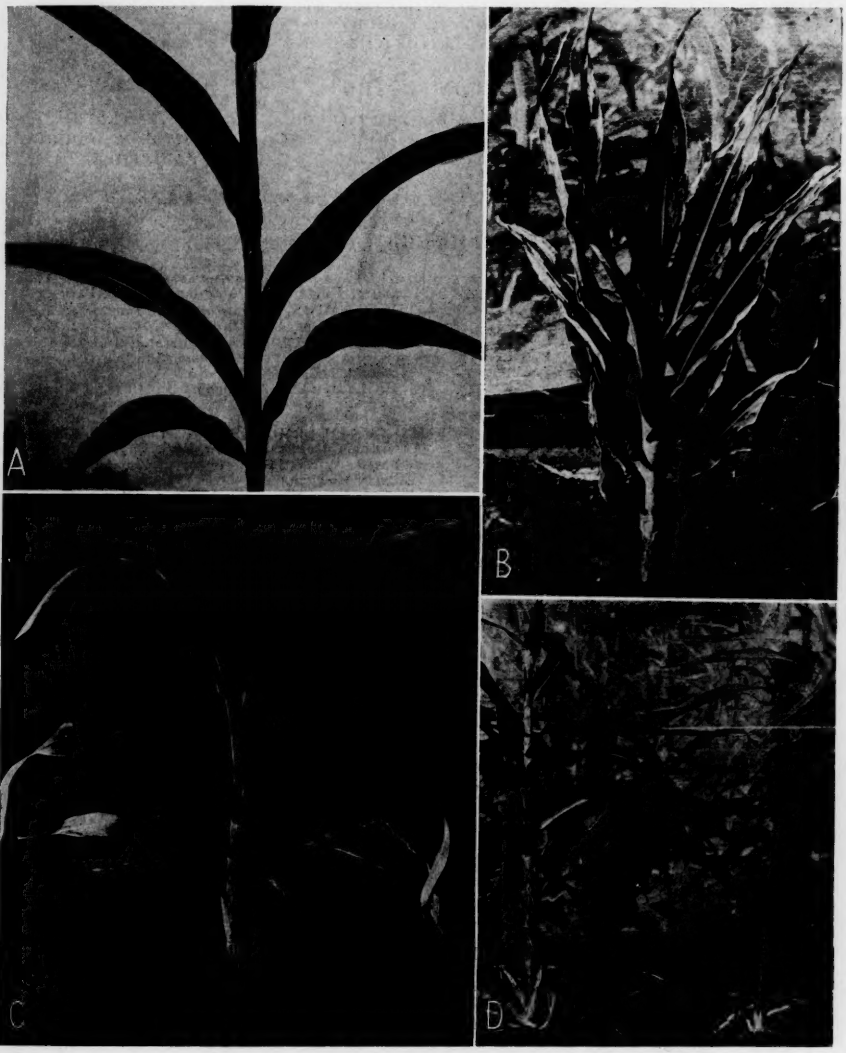

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A Study of Marriage-Customs in North Carolina
Another "Poll-Parrot" Gene in the Fowl
A "Bull-Dog" Mutation in Cattle
A Classification of Quadruplets

VOLUME 33 SEPTEMBER, 1942 NUMBER 9

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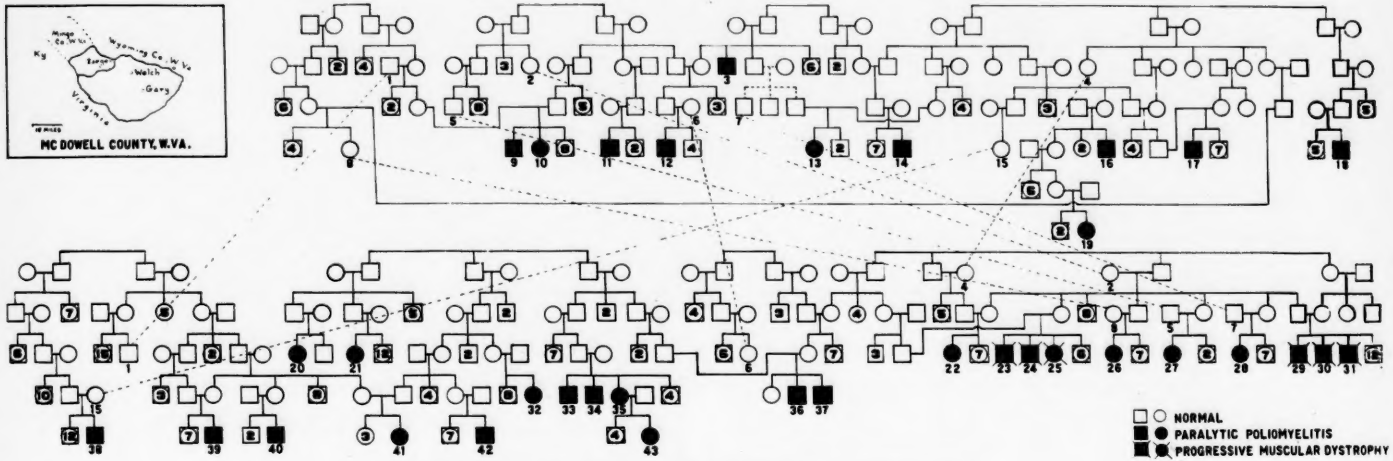

INTERRELATIONSHIPS OF AFFECTED INDIVIDUALS

Figure 1

Pedigree chart of the 29 cases of paralytic poliomyelitis reported in this study. The dotted lines connect the same individual appearing twice in the pedigree. Six cases of progressive muscular dystrophy occurring in these families are also shown. The inset map shows the region of McDowell County, West Virginia, where these cases occurred.

EVIDENCE FOR AN AUTOSOMAL RECESSIVE GENE FOR SUSCEPTIBILITY TO PARALYTIC POLIOMYELITIS

Studies in Human Inheritance XXI

JOHN ADDAIR AND LAURENCE H. SNYDER
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THE occurrence of paralytic poliomyelitis among those exposed to the virus of the disease is infrequent and apparently sporadic. Upon closer examination, however, it is found that the occurrence of paralysis is not as sporadic as may at first appear, but that cases actually tend to cluster in genetically related groups. As long ago as 1898 the work of Taylor[5] suggested a familial tendency. The report of Stephens[4] in 1908 tended to corroborate this view.

In recent years Aycock[1,2,3] has presented a number of family histories which indicate that the major determinant for crippling lies in the host rather than in parasitic factors. Aycock points out that the uniform and wide-spread dissemination of the virus and the equally extensive subclinical immunization in different climates and in inter-epidemic periods suggest that the basis for the genetic susceptibility is a physiologic process which fluctuates with climate and season, rather than an anatomic feature.

In his most recent paper[3] Aycock reviews the earlier literature and summarizes his own data. Nowhere does he venture to suggest the precise nature of the genetic transmission of the factor or factors responsible for the susceptibility. In the present paper evidence will be presented for the existence of an autosomal recessive gene for susceptibility to paralytic poliomyelitis.

Related Cases

The cases of crippling reported in this paper occurred in McDowell County, West Virginia, where the senior author lives and teaches high school. The region covered by this study is indicated by the stippled area of the insert map in Figure 1, and the 29 cases themselves are charted in the figure. These cases represent every occurrence of crippling in the region over a period of 50 years, and are not in any way selected. The ascertainment was thus complete. Medical diagnosis was obtained in each case, largely through the McDowell County Society for Crippled Children. We are indebted to the Secretary of the Society, Mrs. Daisy Early, for aid in obtaining many of the records. The population of the shaded area in the map is 6714, giving an incidence of crippling in this region of one in 231.

In the figure, numbers have been assigned to two sorts of individuals: first, to affected individuals, and second, to those persons who appear twice in the chart. In addition, each person appearing twice has a dotted line connecting his or her two positions on the chart. Inspection of the figure will reveal many interesting genetic relationships of the affected individuals.

There are two instances of two affected sibs each (9 and 10, and 36 and 37) and one occurrence of three (33, 34 and 35). There is one instance of mother and daughter affected (35 and 43). Number 39 is the offspring of first cousins, as is also number 22. Numbers 22, 26, 27 and 28 are first cousins of each other, and are second cousins once removed of numbers 36 and 37. Through another line numbers 36 and 37 are second cousins of numbers 33, 34 and 35, third cousins of number 32, and third cousins once removed of numbers 41 and 42, who are first cousins of each other. Number 32 is a first cousin once removed of numbers 41 and 42. Through another line numbers 39, 40 and 41 are

first cousins of each other, and fourth cousins of number 38.

Numbers 20 and 21 are first cousins, and are second cousins once removed of numbers 32, 33, 34, 35, 36 and 37. Through still another line numbers 36 and 37 are third cousins of number 12. Number 12 is in turn one of a group of second cousins including numbers 9 and 10 (sibs), 11 and 12. These again are all third cousins of the group of first cousins 22, 26, 27 and 28 mentioned above, and numbers 9 and 10 are fourth cousins of number 38, while number 12 is a third cousin of numbers 13 and 14.

Number 17 is the offspring of first cousins once removed, and is himself a second cousin of number 22, and a third cousin of numbers 13, 14, 16 and 18. Through another line number 14 is a first cousin twice removed of number 3. Number 3 is a great uncle of number 12, of number 13, and of number 28, all of whom, as has already been seen, have many affected relatives through other lines. Number 16 is a great uncle of number 19 and a first cousin once removed of number 38.

Other more obscure and complicated relationships between affected individuals may be seen by anyone who cares to trace them out in detail from the figure.

Thus genetic relationships can be traced among all 29 affected individuals. In 14 of these cases (numbers 9, 10, 12, 13, 17, 22, 26, 27, 28, 36, 37, 38, 39 and 41) it has been possible to demonstrate paralytic poliomyelitis both in the father's and in the mother's pedigree, while in 15 of the cases, affected individuals have been demonstrated in the pedigree of only one parent, the other line of descent being unavailable up to the present time. All the cases are alive at present except numbers 13, 26 and 37, who died of poliomyelitis at about three years of age.

Muscular Dystrophy

In the course of this study six cases of crippling were discovered which proved to be progressive muscular dystrophy. These cases are indicated by a special symbol in Figure 1. Numbers 23, 24 and 25 are the offspring of first cousins once removed, while numbers 29, 30 and 31 are the offspring of first cousins. In these two families there were six affected cases out of a total of 24 children, indicating (in these families at least) complete penetrance and constant expressivity for the recessive autosomal gene for progressive muscular dystrophy. Both sex-linked and autosomal forms of this gene have been previously recorded.[6]

Evidence for Recessive Gene

The pedigrees of paralytic poliomyelitis herewith presented provide good evidence for the existence of a recessive gene for susceptibility, and an examination of Aycock's pedigrees[1,3] bears out this claim. However, frank paralysis is not found in a quarter of the children in families where one is affected, but in one out of 5.5 (29 out of 161). Actually we should expect somewhat more than a quarter of the children to show the paralysis in families where one is affected, because in a certain proportion of matings of heterozygous parents no affected children will by chance appear although in fact they could occur. In a random-breeding population this correction can readily be made, but in interrelated families such as these the computation becomes involved. Yet a simple recessive autosomal factor appears to be the most reasonable explanation of these families. Considered from the standpoint of frank paralysis, we may say that the gene shows reduced penetrance. Since we should expect somewhat more than 40 cases in these families, and obtained but 29, we may roughly estimate the penetrance at 70 per cent.

A possible explanation for the reduction in affected cases lies in the early age of onset in this group. Children born in inter-epidemic periods may pass the most susceptible age before exposure. This point is being further studied.

On the other hand, we know that a range of effects from frank paralysis through milder and non-paralytic cases to abortive infections occurs, and from this standpoint we might suspect that

the gene for susceptibility shows variable expressivity, resulting in frank paralysis in 70 per cent of the homozygotes, and milder infections which pass almost unnoticed in 30 per cent. An attempt was made to determine the occurrence of fevers or other illnesses in the families during the onset of paralysis in the affected members. In no case was any normal sib apparently even mildly ill. It would therefore appear that sub-clinical cases, where found, are variable expressions of the dominant gene for immunity, rather than of the recessive gene for susceptibility.

The onset of paralysis in these cases occurred during the first two years of life. The affected individuals include 16 males and 13 females, a non-significant deviation from a 1:1 ratio.

The terrain of the region is extremely rugged, being broken up into mountains traversed by narrow winding valleys. The various families involved in the chart are thus more separated geographically than would be the case in flat country. Although a certain amount of inbreeding and intermarriage is apparent in the families presented here, the community as a whole is not highly inbred. The families containing affected cases live in many different valleys, along different streams, and are often completely out of touch with one another. There are hundreds of families living among those shown in the figure in which no trace of paralytic poliomyelitis has occurred. Here, as elsewhere, the affected individuals tend to cluster in genetically related groups.

It is obvious that many questions important from the standpoint of epidemiology are raised by this study. A careful survey from this point of view is now in progress and will be reported in a later paper.

Summary

Twenty-nine cases of paralytic poliomyelitis, involving complete ascertainment in the northwest portion of McDowell County, West Virginia, are presented in family-history form. The genetic interrelationships of these cases suggest the existence of an autosomal recessive gene for susceptibility to paralytic poliomyelitis, showing about 70 per cent penetrance.

Literature Cited

1. Aycock, W. L. *West Va. Med. Jour.* 30:481. 1934.
2. ———. *Ill. Med. Jour.* 80:231. 1941.
3. ———. *Amer. Jour. Med. Sci.* 203:452. 1942.
4. Stephens, H. D. *Intercol. Med. Jour. Australasia* 13:573. 1908.
5. Taylor, J. M. *Philadelphia Med. Jour.* 1:208. 1898.
6. Weitz, W. *Deutsche Zeitschr. f. Nervenheilk.* 72:143, 1921; 102:209, 1928; 107:316, 1929.

✦

Breeding Farm Animals

KNOWLEDGE of genetics and physiology of reproduction and of the applications of these sciences in animal breeding practice have increased rapidly in recent years. Accounts of many of the recent advances have been incorporated in the third edition of Rice's text.* It is a more comprehensive volume than the second edition and the weaknesses which Warwick† pointed out have been largely corrected.

The student is introduced to the subject of animal breeding by a statistical and historical review of the livestock industry and a discussion of the possibilities in livestock breeding and improvement. This introductory section is well written and should give the student an appreciation of the background of modern animal breeding and of the immense progress man has made, both in the art of thinking and in the art of breeding better livestock.

The second section deals with the

*Rice, Victor Arthur. Breeding and Improvement of Farm Animals. Pp. 750. Third edition. Price $5.00. The McGraw-Hill Book Company, New York. 1942.

†Warwick, B. L. Breeding Farm Animals. *Jour. Heredity* 26:243-245. 1935.

mechanisms of reproduction. This section has been materially strengthened by the addition of chapters on the endocrine basis of reproduction and lactation and on artificial insemination.*

The third section deals with the mechanism of heredity and is a well-organized review of the achievements in fundamental genetics from the time of Mendel to the present. This leads to the fourth section, "The Art of Breeding. This might better have been called "The *Science* of Breeding" since it represents an attempt to bring together the results of research and to show how they can be applied to make animal breeding a *science* rather than an *art*. The various systems of mating are discussed and particular attention is given to inbreeding, and the possibilities of livestock improvement through its use. The author points out that "Inbreeding will be successful (1) if the good genes pretty well outnumber the bad ones to start with, (2) if the breeder has more than average skill in planning his matings, (3) if the breeder can and will practice very rigid selection. Finally, it isn't the system of mating—it's the genes in the animals and the intelligence in the man, and the latter is also largely a matter of genes."

The chapter on selection in dairy cattle is particularly comprehensive and well written. The chapters on selection in horses and the meat animals do not measure up to the one on dairy cattle, but this is a reflection of the generally inadequate knowledge in this field rather than any lack of effort on the author's part. The thoughtful student should recognize that much must yet be learned before selection in work and meat animals can be placed on an objective basis and the records of performance put to real use in breeding practice to the extent they can now be used in dairy cattle. The author points out that "The greatest 'lack' in breed associations today is the lack of records of performance. A pedigree that consists merely of names of ancestors without production performance of the ancestors and collaterals is of little value. If, however, the pedigree could be made more complete in terms of records or characteristics, it would be an invaluable guide in the selective mating of animals."

The author writes in an interesting style, and his frequent attempts to look into the future should stimulate the reader to delve deeper into the many problems introduced in the text. The close relationship between man and his animals is kept constantly before the reader and is well illustrated by the closing lines of the book:

> A proton and electrons, then elements emerge—
> They organize and synthesize to form a living surge.
> Then animals and man appear in orderly progression:
> Teamed, they move with quickened pace—a fitting, joint possession.

RALPH W. PHILLIPS

Bureau of Animal Husbandy, Beltsville, Md.

*These two chapters were written by Frederick N. Andrews of Purdue University, Lafayette, Indiana.

TYPES AND FREQUENCIES OF QUADRUPLETS

Studies of Quadruplets — III

H. H. Newman and Iva C. Gardner

THIS paper and others to follow are intended to be parts of a more extensive study of multiple human births other than twins. Since only one set of quintuplets exists, our present and future reports must of necessity confine themselves to quadruplets and triplets. This and the next few reports deal with quadruplets.

Actually, since surviving whole sets of quadruplets in the United States are rather rare, it has seemed to us worth while to make an effort to study and report every case that we can discover within the confines of this nation. Hence one of the objects of the present paper is to arouse interest in quadruplets in the hope that any cases possibly overlooked by us may be brought to our attention.

After extensive inquiry we have been able to locate only seven surviving whole sets of quadruplets in the United States. We have already studied and reported in this Journal[1,2] our analyses of two sets, the Perricone quads (a four-egg set, all boys) and the Keys quads (a three-egg set, all girls). We have now on hand data on four additional sets of quads, the analyses of which we shall present case by case. Last year we had eight sets of quads on our list, but since then one set born in North Dakota has been broken up by death.

As in previous reports by the present authors, Dr. Gardner has done the travelling about the country collecting the data and Dr. Newman has taken the data as given him, studied and analysed them, diagnosed the zygotic origin of the sets, and in consultation with Dr. Gardner, has prepared the paper for publication.

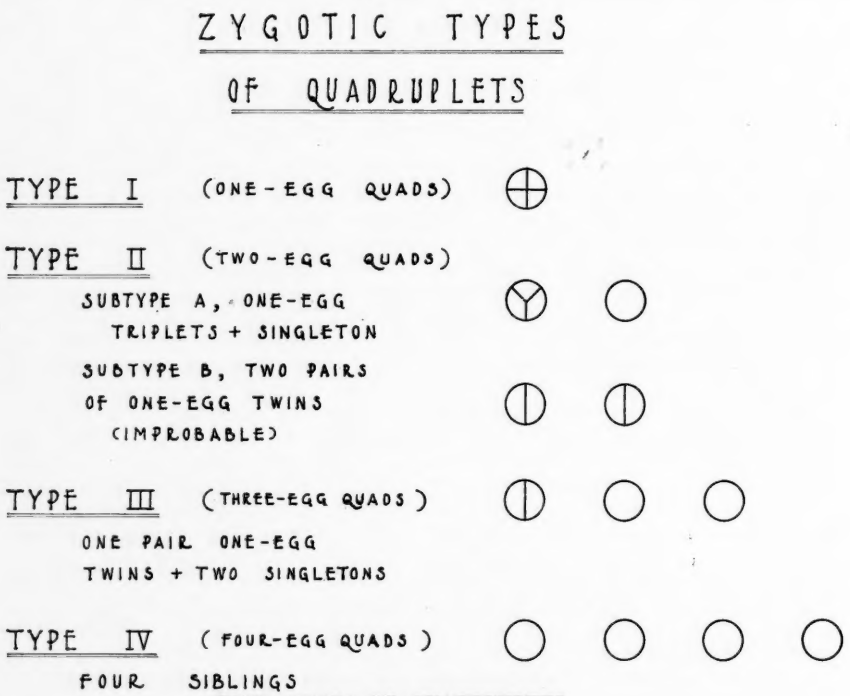
Kinds of Quadruplets

As is well known to students of multiple human births, there are only five theoretically possible distinct kinds of quadruplets, when their modes of zygotic origin are considered: one-egg quadruplets; two kinds of two-egg quadruplets (*a*, a set of one-egg triplets plus a "singleton," and *b*, two pairs of one-egg twins); three-egg quadruplets, consisting of a pair of one-egg twins plus two "singletons"; and, finally, four-egg quadruplets. These five types may be readily visualized by following the accompanying diagram (Figure 2), in which each circle represents a single egg, or zygote, and the sections of the circle represent the products of one-egg twinning.

We now have in the collection of quadruplets that have already been studied representatives of four of the five zygotic types illustrated in the diagram. As yet we have not discovered nor even heard of a case of the second type of two-egg quadruplets, namely, that consisting of two pairs of one-egg twins. For the sake of completeness we would be especially pleased to obtain information about a set of this type, though we have reason to regard the occurrence of this type as highly improbable. Why we hold this opinion will now be explained.

If, on the one hand, one-egg twinning be hereditary, it seems highly improbable that any two zygotes ovulated simultaneously would both receive the combination of genes necessary for one-egg twinning, for at best the genetic basis for one-egg twinning must be somewhat complex. Otherwise the hereditary pattern seen in twinning pedigrees would be much simpler than it is and more in accord with known Mendelian ratios.

If, on the other hand, one-egg twinning be environmentally induced either by delayed placentation or by underactivation by defective sperm, it seems unlikely that two zygotes, genetically different, would be simultaneously and equally affected in such a way as to produce two pairs of one-egg twins both



FOUR TYPES OF QUADRUPLETS

Figure 2

Diagram illustrating all possible zygotic origins of quadruplets. Each circle represents a fertilized egg (zygote); and the sectors of circles individuals resulting from the fission-process which produces one-egg twins. The double-identical twin subtype of Type II represents the simultaneous occurrence of both types of twinning, an event so rare that it makes the occurrence of this type of quadruplets very unlikely.

sufficiently viable to develop to term and be born alive in a vigorous, healthy condition.

For these reasons we are far from hopeful of ever discovering an example of our Type II-b quadruplets. It might be well, therefore, to eliminate that type from our list and consider that there are only four zygotic types of quadruplets, one-, two-, three-, and four-egg types, and only one kind of each type. In our present collection all of these types are represented. The Morloks are a one-egg set, the only one in existence that we know of; the Kaspars and Badgetts are both, according to our diagnoses, two-egg sets; the Keys are a three-egg set; and both the Perricones and the Schenses are four-egg sets.

Classes of Quadruplets According to Sex Distributions

In addition to these four primary, or zygotic, types of quadruplet sets, there are four times as many kinds of sets when the various premutations of the sexes within the set are taken into consideration. These may be listed as follows:

TYPE I (one-egg quads)
1, four males
2, four females
TYPE II (two-egg quads)
3, four males
4, four females
5, three males and one female
6, one male and three females
TYPE III (three-egg quads)
7, four males
8, four females

9, three males and one female
10, two males and two females
11, one male and three females
TYPE IV (four-egg quads)
12, four males
13, four females
14, three males and one female
15, two males and two females
16, one male and three females

The first six sets of American quadruplets thus far studied and diagnosed by us are classed as follows, giving each its number according to the above list of sex-distribution types:

The Morloks (2)
The Badgetts (4)
The Kaspars (5)
The Keys (8)
The Perricones (12)
The Schenses (15)

The remaining set of which we have knowledge, the Lashley quads, are still too young for satisfactory diagnosis and their classification will have to be postponed for a later report. All that we can say at present is that the set consists of one boy and three girls, which would place it in one of three categories, 6, 11, or 16 according to whether it turns out to belong to Type I, II, or III. So, from the standpoint of sex distributions within the set, we have so far no duplicates.

It will be noted that four of the seven sets are same-sexed, one all males and three all females. Thus 57 per cent of this small collection of quadruplets are same-sexed. Among the 48 sets of quadruplets born in the United States between 1915 and 1930 Hamlett[3] found that 23 (48 per cent) were same-sexed, 13 sets being males and 10 females. If our seven sets be added to Hamlett's collection almost half of the total number of quadruplets turns out to be same-sexed. At first sight these data might seem to suggest that the majority of the same-sexed sets are monozygotic, but other considerations render this suggestion quite erroneous. In the first place, only one of the four same-sexed sets in our collection can possibly be diagnosed as one-egg quadruplets. In the second place, an examination of the sex probabilities of two-, three- and four-egg quadruplets sets renders the incidence of same-sexed sets in each of these types higher than at first might be expected.

Assuming, as we do, that there is only one type of two-egg quadruplets, namely, that consisting of one-egg triplets plus a "singleton," it is obvious that there is an even chance that the "singleton" will be of the same sex as the triplets, and that half of such sets will be same-sexed.

Among three-egg quadruplets the expectation is that one-fourth of the sets will be same-sexed, and among four-egg sets the expectation is that one-eighth will be same-sexed. Summing up, then, it will be seen that all one-egg sets, half of the two-egg sets, one-fourth of the three-egg sets, and one-eighth of the four-egg sets should be same-sexed. If the four zygotic types occur with equal frequency and the above expectations as to the incidence of same-sexed sets holds, about 47 per cent of all quadruplet sets should be same-sexed, which is very close to the figure determined by Hamlett for the 48 sets which he studied. This might also be taken as supporting evidence that the four zygotic types of quadruplets actually do occur with approximately equal frequency. Moreover, in our small collection of quadruplets there are four same-sexed sets, one of each of the four zygotic types: the Morloks a one-egg set, the Badgetts a two-egg set, the Keys a three-egg set, and the Perricones a four-egg set. This might be taken as further evidence that the four zygotic types occur with approximately equal frequency.

Sex Distribution

Another rather odd feature about the sex ratios in Hamlett's collection of quadruplets that has caused considerable comment is the rather high preponderance of males over females, 101 males to 91 females, a ratio that agrees rather closely with that determined by MacArthur and Ford[4] for the 40 sets of quintuplets for which the sexes had been recorded, namely, 110 males to 90 females. Various rather far-fetched theories have been offered to account for this male preponderance. The significance of these aberrant sex-ratios is cast into con-

siderable doubt by the fact that when we add our seven sets to Hamlett's 48, the sex ratio of the whole 55 sets is 111 males to 109 females, a little lower preponderance of males than that in the general population. We would be inclined, therefore to suspect that the alleged excessive male preponderance in multiple human births is illusory and that speculations as to its significance are premature in view of the paucity of data.

This brief survey of quadruplets in general may be taken as a general introduction to a series of reports on the individual sets which we hope to publish later in this JOURNAL.

Literature Cited

1. GARDNER, IVA C., and H. H. NEWMAN. *J. Hered.* 29:(9). 1938.
2. ————————. *J. Hered.* 31:(4). 1940.
3. HAMLETT, G. W. D. *Genetics* 20: 1932.
4. MACARTHUR, J. W. and NORMA FORD. Chapters in Collected Studies of the Dionne Quintuplets. Univ. of Toronto Press. 1937.

Medical Genetics—the Alice-in-Wonderland Way*

THIS book sets out with a worthy but ambiguous purpose: "to clarify the origin of hereditary and somatic disease, and in general to enumerate the foods and methods used in preventing and curing them." The general point of view is one which deserves circulation—that since children inherit differences in constitution, and acquire differences through the conditions of their lives, "no two children can be raised alike, no two nourished alike . . . no two can be treated alike medically, although suffering from the same malady. . . ."

The present reviewer is not technically qualified to appraise the *medical* sections of the volume—the 700 pages more or less devoted to the diagnosis and treatment of disease, and the role of nutrition in both. Throughout these 700 pages, however, constitutional or hereditary factors come in for frequent mention. Where they do, the treatment is consistent with that in earlier chapters on "Heredity and Environment" and "Phenomena of Mating." The author conveys such a fantastic and confusing conception of the principles of heredity as the writer has never before seen in any textbook.

It will be of interest to cite some of the statements which Dr. Scott evidently expects us to accept, either with no documentation at all, or with citation to outmoded investigations. It is a fact that none of the literature citations in the chapter on "Heredity and Environment" is of later date than 1927.

Concerning "vigor," defined as a "state of high metabolism" the author has this to say: "This united vigor in the parents depends naturally on their rate of metabolism at the time of copulation, and its rate will depend upon their initial vigor modified by their environment. If two parents have the same vigor, their offspring will be of that vigor. Also, if the vigor of both these parents be raised to the same extent by favorable conditions, their second offspring will have a higher initial vigor than the first. (p. 3) . . . The differences of power in fertility which is passed on to succeeding generations must be greatly, if not entirely, due to the vigor of the parents." (p. 4)

That "vigor" and "age and training of parents" affect the offspring are two popular superstitions whose vigor has been imperceptibly reduced by a total lack of experimental or observational evidence. The reviewer has never seen these hoary myths more vigorously propounded, and never under more deplorable circumstances.

On mode of transmission: ". . . In the higher animals heredity can be recognized only as a result of some physiologic process. True, the inherited character is not of itself a physiologic one, but is a

**Heredity, Food, and Environment in the Nutrition of Infants and Children.* By GEORGE DOW SCOTT, M.D. Pp. xvii+778. $5.00. Chapman and Grimes, Boston, 1942.

structure produced by some physiologic activity, a circumstance which is not always true in the lower animals. In the higher animals, the humans, the characters are handed from parent to offspring fully formed and in the same condition in which the character will appear in the infant when it becomes an adult, for the completeness of characters is only seen in maturity. In the higher animals, however, there is never any such direct transmission without a change from one generation to another." (p. 5)

To requite for difficulties of parsing the grammatical structure, and of a biological parsing of completely contradictory statements, the reader can concoct his own ribald wisecracks along the line of: "Pappy, pass the liver!"

The author places Galton, whose pioneer study *Hereditary Genius* was published in 1869, in the late seventeenth or middle eighteenth century. (p. 6)

"The phenomenon of dominance is an unessential feature of Mendelian inheritance, for we are almost all hybrids of one kind or another." (p. 10)

Such a statement completely misses the central issue in any eugenic program aimed at the elimination of hereditary defects, which is to prevent reproduction by people with dominant defects and (if they can be identified) by carriers of recessive defects.

"The chromosomes are the bearers of hereditary units and many such units are carried by each chromosome. We might assume, by way of illustration, that the heart, liver, lungs, kidneys, etc., are all from one unit." (p. 19)

The reviewer finds it fascinating to assume by way of additional illustration —or fantasy—a gene allelic to Scott's heart-liver-lung-kidney gene, which would determine temperament-digestion-excretion-reproduction! [Editor: How about having liver allelic to onions, but then pappy couldn't pass both, which would be deplorable.]

"It may happen in cell division that all the maternal chromosomes go to one cell and all the paternal to another, the child resembling the mother or father in an amazing mass of characters and traits." (p. 21)

The only phrase which occurs to the reviewer as adequate to dispose of this innocuous-sounding but portentous sentence is: "immaculate misconception"!

"Linkage insures that more than 25 per cent of the offspring will exhibit the same combination of characters as was possessed by one of the grandparents, and that more than 25 per cent will be like the other grandparent. This is known as absolute linkage, and fortunately for many children, is not common. Some children, however, take one character from one grandparent and another character from the other grandparent. This, as already stated, is called crossing-over, and is a form of arrangement which establishes a new linkage. . . . The striking resemblances of children to one parent in a number of ways may be due partly to linkage rather than to a more random combination of hereditary factors." (Pp. 24-25)

This might work with Scott's gene for heart-liver-lungs-kidneys or for the reviewer's suggested allele, but hardly with the genes which are reported in the literature of human (or more lowly) genetics.

"Some outstanding figures in history were the result of intensive inbreeding. Among them were Peter the Great of Russia, King Ferdinand, Queen Isabella, and Charles V of Spain, Frederick the Great of Prussia, Gustavus Adolphus, and Charles XII of Sweden. In truth, the concentration of ability due to fortunate royal matings decided their characters, yet their transmitted genius was dissipated later through unwise unions. History tells us that almost without exception the descendants of these individuals were epileptic, mediocre, imbecile, feeble-minded, immoral, cruel, or depraved, — all recessive characters transmitted to the males in line, while the females escaped." (p. 43)

This paragraph rewards careful study by geneticists as well as by historians.

Mediocrity is indeed travelling in fast company in the midst of such a galaxy of wicked recessive genes, all apparently located on the Y-chromosome!

These examples of genetical infelicities could be multiplied many times, but there would be little point in doing this, even for collectors, since the elusiveness of genetical principles to the book's author is amply demonstrated. The volume revives in the reviewer's mind an ever-recurrent question: why it is that certification in one of the "learned professions" should give the certificate-holder — and publishers of books — cause for belief that he is competent to sound off as an authority on fields both near to and remote from the field of his own training and practice? The book also underlines a fact which the faculties in modern schools of medicine are more and more realizing: that genetical training has been sadly lacking in the medical curriculum; that the physician who has any grasp of the principles of medical genetics is the rare exception. The successors of Hippocrates can no longer afford to neglect one of the most essential branches of their "armed services." The author of the book knows this too, but he is evidently not one who can remedy this lack in the training of his young colleagues.

At the risk of causing a multitude of mental conflicts the reviewer wishes to go on record that hardly anywhere else can be found as many genetic belly-laughs for five dollars. To reward the publishers by making a best-seller out of the book for this reason would be unfortunate. Perhaps it is just as well that not many geneticists will be in the mood to sink a fiver merely for fun in so grim a world. A modest reward for Messrs. Chapman and Grimes may be justified, for one can even forget Stalingrad when Dr. Scott takes us by the hand and leads us through the garden of the genes.

BARBARA S. BURKS
Columbia University

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Research on Twins with Mental Disease

The division of genetics research, in the department of psychiatry, was again particularly active throughout the year. Another grant from the Carnegie Corporation of New York, on the basis of the generous recommendation of Mr. Frederick Osborn, made it possible for Dr. Kallmann to continue the study of psychotic and tuberculous twin pairs. Under the terms of the grant, the first six months were used predominantly for completing the collection of the clinical and genealogical data on all the twin cases included so far in the study, and for bringing all of this material up to date. During the second half of the year, the work was concentrated in preparing the main surveys of schizophrenic, manic-depressive and mentally-defective twin pairs for statistical analysis and publication (in cooperation with Dr. Lowell J. Reed of Johns Hopkins University School of Hygiene and Public Health and Dr. Paul R. David of Allentown, Pa.). During the year it was possible to enlarge the total twin material by 252 pairs, of which 101 were psychotic, 107 were affected by active pulmonary tuberculosis and 44 were tuberculosis contact cases with definite exposure to a tuberculous family case. The present total material of twin pairs available for analysis consists of 1,317 pairs of which 618 are psychotic twin pairs, 301 tuberculous twin pairs, 152 tuberculous contact twin pairs, 34 special sets of twins and triplets with various anomalies and 212 normal twin pairs in older age groups.

The visits to the homes of twin families and to those 154 mental hospitals, State schools and tuberculosis sanatoria, among which the newly reported twin patients were distributed, required driving over 10,000 miles.

The present status of the various twin surveys is as follows:

The first analysis of a group of 53 twin pairs with mental deficiency in one or both members was completed during the past year and was published in the American Journal of Mental Deficiency, Vol. XLV, No. 1 and 4. An additional report on several special twin pairs affected by mongolian idiocy will be completed and published in the same journal within the next months.

The collection of the clinical and genealogical data on 412 schizophrenic twin pairs and their families has practically been completed and will be ready for statistical analysis and publication very shortly, together with an anthropometric analysis of the somatotypes of a special group of concordant schizophrenic twin pairs and studied at present at the Institute according to the newly-developed Sheldon

(*Continued on page* 327)

A NEW TYPE OF RECESSIVE ACHONDROPLASIA IN CATTLE

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An achondroplastic or "bull-dog" type of sub-lethal hereditary defect has occurred in the Jersey herd of the California Experiment Station. Achondroplastic hereditary types have been known in cattle for a considerable time, but this defect differs in certain respects from those preivously reported.

In the past few years several authors (Hutt,[6] Eaton,[5] Mohr[7,8]) have reviewed the literature of hereditary defects of domesticated animals. No attempt will be made here, therefore, to review the general literature of lethals, but it will be necessary to consider somewhat similar achondroplastic types in cattle.

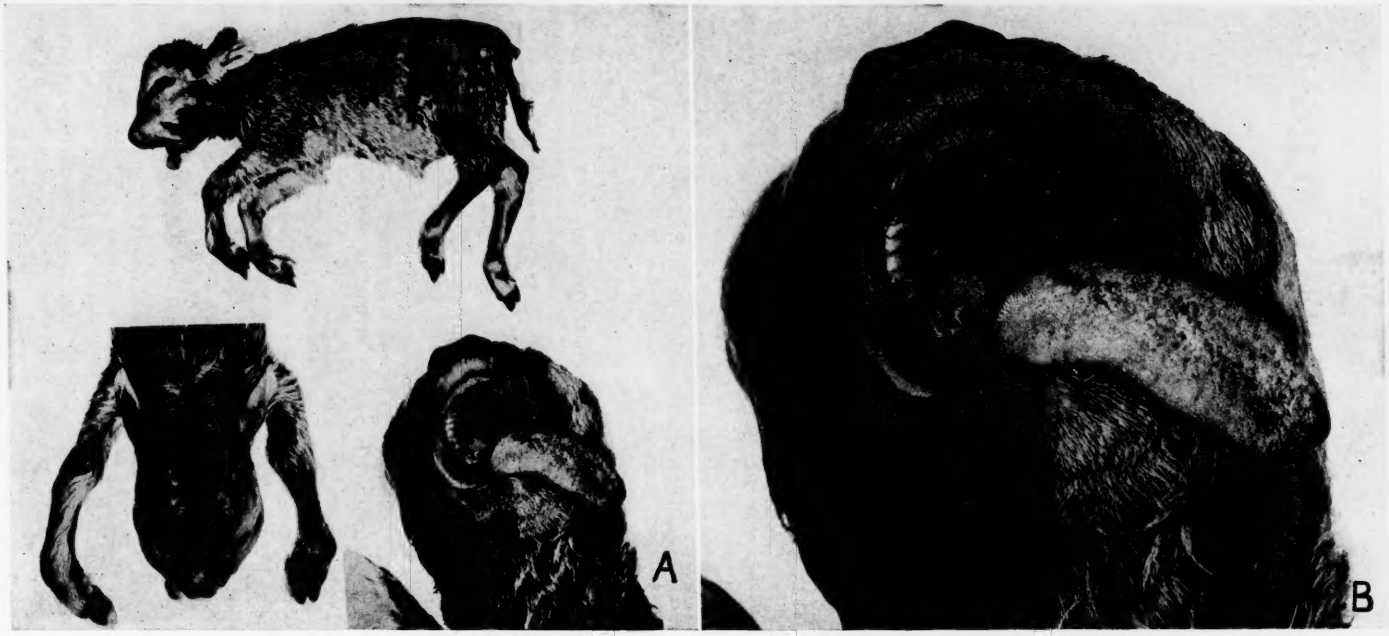
One achondroplastic type is conditioned by a dominant (or semi-dominant) gene, lethal in the homozygous state. This defect has been studied by Crew[3] and other investigators. It is found in the heterozygous state in Dexter cattle and causes a shortening of the legs. When Dexters are mated together the progeny are of three types: one-fourth are homozygous dominants, exhibiting marked achondroplasia, and are aborted during the latter part of the gestation period; one-half are of the heterozygous Dexter type with shortened legs; and the remaining one-fourth are homozygous normal.

Wriedt[10] and Mohr[7] have found a somewhat similar, yet genetically different, achondroplasia in the Telemark breed of Norway. This defect is inherited as a simple recessive. Homozygous normal and heterozygous animals are phenotypically alike. The defective calves are born alive, but die within a few days because of a respiratory paralysis caused by their inability to stand. All the anomalies observed are very uniform in type. Both axail and appendicular skeletal structures are affected. Diagnostic characters are a shortened head, deformed jaws, a cleft palate, and short stumpy legs. According to Eaton,[5] a similar type of anomaly has been reported in European Holsteins and Downs[4] has observed it in grade Holsteins in the United States. Carmichael[2] reported achondroplasia in Nganda cattle which Punnett[9] regards as the Telemark type. Brandt[1] reported achondroplasia in grade Guernseys and grade Jerseys. This achondroplasia, recessive in nature, is presumably identical with the recessive Telemark type.

A recessive achondroplastic sub-lethal defect resembling somewhat the one described by Wriedt[10] and Mohr[7,8] appeared in the California Experiment Station herd in 1927 (Table I). Even though the defective calf was somewhat inbred (Figure 4, pedigree *A*) and a genetic basis was suspected, no hereditary basis was established until several years later when additional defective animals were produced. The second defective calf, a male, was born February 14, 1933; and about 13 months later the same sire and dam produced a second defective animal (Table I and pedigree *B*). Sire 433*A* produced six additional achondroplastic calves in the California Experiment Station herd when he was bred to his own daughters or to his half sisters. Two additional defective calves were produced in herds of cooperating dairymen who were using bulls descended from either 320*A* or his inbred son 433*A*.

Description

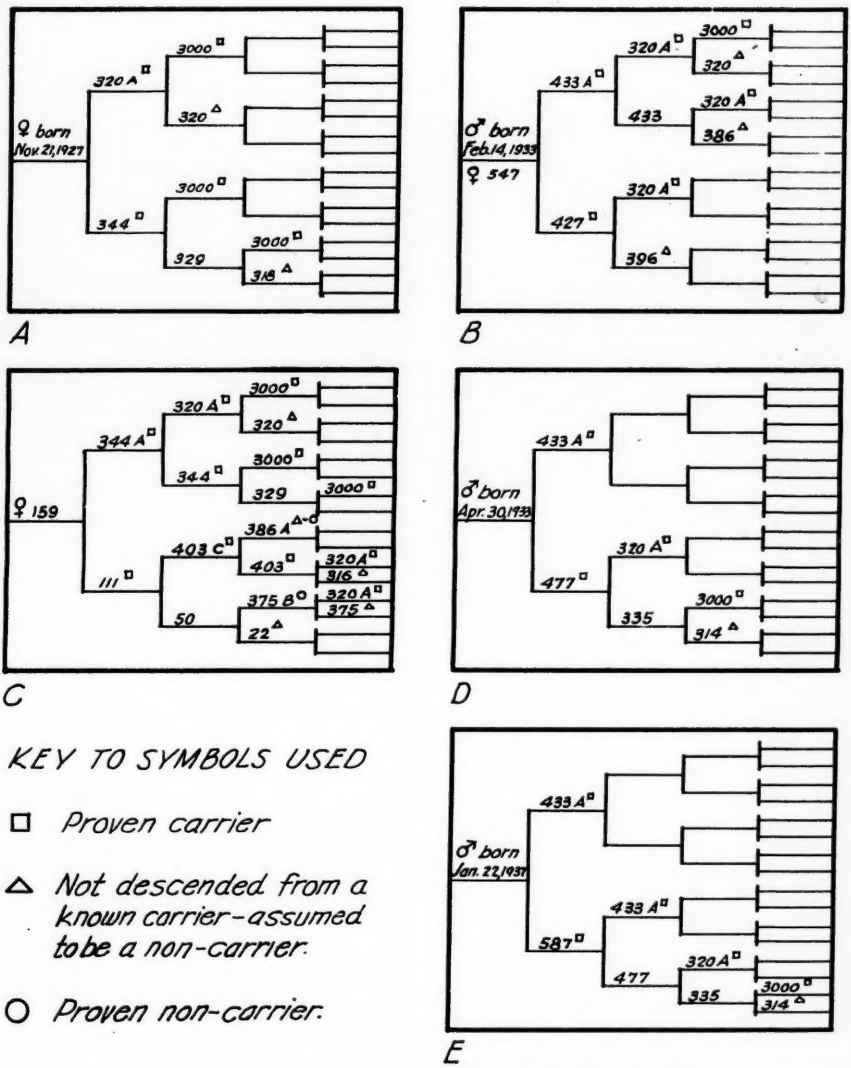
This defect is extremely variable in its expression. Usually, but not always, it is lethal. Both axial and appendicular skeletal structures may be affected. One of the most extreme types, a female (Figure 3) sired by 433*A* and born Jan-



RECESSIVE ACHONDROPLASIA

Figure 3

A—Three photographs of the defective female calf born January 8, 1935. Compare with description in text and with recessive types of achondroplasia reported by other investigators. *B*—Enlarged view of mouth and head.



PEDIGREES OF CERTAIN ACHONDROPLASTIC CALVES

Figure 4

A—The first defective calf born. *B*—Bull calf born February 14, 1933, and heifer calf born March 25, 1934. *C*—Heifer 159. This pedigree, coupled with other pedigree studies and progeny tests, illustrates how the absolute or probable genotype of individual animals may be determined. (See text.) *D*—Somewhat similar to pedigree *A* except that a slightly different type of inbreeding is used. *E*—Three carrier bulls appearing in the first four generations of a calf born January 22, 1937. All three herd sires were selected by chance so far as the achondroplastic gene is concerned.

uary 8, 1935, from dam 480, was autopsied. The following description was taken from the autopsy protocol.

Gestation period 287 days, normal. No one present at birth. Still-born or died soon after birth. Size about normal. Head deformed, short and broad. Ears short, 3.5 inches. An unclosed large fontanel due to failure of the cartilage to become ossified. Lower maxillary bone present, but greatly deformed. Six incisor teeth erupting in an irregular manner, giving an irregular surface.

Lower maxillary symphysis is extremely short and not ossified. Chief anomaly of this bone is a foreshortening of the rami (3 inches) so that the incisor teeth meet the upper jaw opposite the first molars instead of coming into apposition to the dental pad; thus the tongue, which is normal in length, hung loose over the incisors. Extreme degree of cleft palate. Failure of closure of the superior maxilla, leaving an incomplete hard palate. Nasal septum exposed to the mouth cavity. Marked foreshortening of the superior maxilla, though not to the extent observed for the lower maxilla. Development of the hard palate about half an inch wide on each side (edge) of the upper jaw, but the open space between the inner edges is about two inches wide and is occupied in part by two maxillary turbinates which appear truncated. Molar teeth are present on both upper and lower jaws.

Length of legs not appreciably reduced. Suspensory ligaments were holding the anterior pastern joints in marked flexion. Pasterns assumed normal position and motion when the ligaments were cut.

The least extreme case was female 159, born in the herd of one of our cooperators (pedigree *C*, Table I). When observed at six months of age, this calf had a short, broad head and legs that were slightly reduced in length. She was considerable below average size, and this difference became more pronounced with age. After having been kept in the herd almost 14 months, she was sold for slaughter. She had a fair amount of vigor, but less than that of her normal half sibs. She was observed to be in estrum at about 8 months of age.

All the other calves exhibiting this anomaly varied between the two extreme types described. All possessed the short, broad head that is quite diagnostic. Cleft palate is common, though not always present. Length of legs may be slightly reduced. It might have been possible to raise some of the other calves showing the defect in a mild form, but only one attempt was made. Evidently this defect is quite variable in its expression.

Inheritance

Since the defect is usually lethal in both sexes and was uncovered by inbreeding normal animals, one would suspect that it was inherited as an autosomal recessive. All the matings in the University of California herd that would permit defective animals to segregate in the progeny can be classified into three types: (1) the mating of a carrier bull to his own daughters or the daughters of another carrier bull as in pedigree *B*; (2) the mating of a carrier bull to daughters and granddaughters of carrier sires as in pedigree *D*; and (3) the mating of a carrier bull to daughters, granddaughters, and great-granddaughters of carrier sires as in pedigree *E*. Table II summarizes the results from each of these matings, besides indicating the appropriate calculated expectancy and the deviations for each type of mating. In each type of mating the sex ratio is approximately equal in both the normal and defective classes, and the observed results approach rather closely the expected on a monofactoral autosomal recessive basis. It is concluded, therefore, that this type of achondroplasia is conditioned by one recessive autosomal gene.

Origin of the Mutation

The first achondroplastic calf that appeared was sired by 320*A* (pedigree *A*), and most of the succeeding ones were sired by his inbred son, 433*A*. Defective calf 159 was sired by bull 344*A*, another son of 320*A* out of an inbred daughter of the sire of 320*A*. Another defective calf was produced by mating 403*B* to his own daughter. This sire is a son of 433*A* out of a daughter of 320*A*. Bull 320*A* was the sire, grand sire, or great-grand sire of all the achondroplastic calves born. As pedigree *A* indicates, however, the original mutation did not occur in sire 320*A*. Sire 320*A* and cow 344 evidently obtained the mutant gene from their common sire 3000. The trail ends with sire 3000, but it would be interesting to know whether or not the muta-

tion occurred with him or originated further back in his ancestry. This type of defect may be carried in the heterozygous state for an indefinite period without coming to light.

Since the breeding program of the Experiment Station Jersey herd is primarily an inbreeding one designed to fix, in a more homozygous state, the factors for high milk production, the genotype relating to this defect has been determined in a relatively large number of sires and dams. By use of animals of proved genotype, coupled with pedigree studies, an unbroken chain of carrier animals extending through several generations may often be identified. The mildly defective calf, number 159, born in the herd of one of our cooperators, is an excellent illustration (pedigree *C*.). Until the birth of this calf the only proved carriers appearing in pedigree *C* are 320*A*, 344, and 3000 (from pedigree *A*).

Upon the birth of 159, sire 344*A* and cow 111 became proved carriers. The next object is to determine, if possible, the path by which cow 111 obtained the mutant gene. Cow 111 has equal possibilities of obtaining the gene from her sire or dam, since 320*A* is both a paternal and a maternal great-grandsire. All animals not descended from 320*A* may be eliminated as a probable source of the gene. Bull 386*A* produced sufficient progeny from his own daughters to prove that he is a noncarrier, as he was assumed to be. Sire 375*B* is a potential carrier, being a son of 320*A*. Test matings to his own daughters proved him, however, to be a noncarrier. Since cow 22 is a foundation cow in this herd and is unrelated to any of the carrier bulls, the chance of her being a carrier is negligible. Cow 403 has a probability of 0.5 of carrying the gene. Evidently the path by which cow 111 obtained the gene is through 403 and 403*C*. This type of analysis is often a useful aid in determining the absolute or probable genotype of individual animals in a pedigree. This defect is being eliminated from the herd by the use of proved homozygous normal bulls.

From the description and the mode of inheritance it is clear that the achondroplastic type herein reported differs from the dominant type found in Dexter cattle. Judging from description and inheritance, it somewhat resembles the recessive type of achondroplasia first reported in Telemark cattle of Norway. When the description and photographs of the cases reported by Mohr,[8] Downs,[4]

TABLE I. A Tabulation of Achondroplastic Calves Produced in the Jersey Herd

No.	Sex	Date born	Sire	Dam	Dam's sire
----	Female	11/21/27	320A	344	3000
----	Male	2/14/33	433A	427	320A
----	Male	4/30/33	433A	477	320A
540	Female	8/8/33	433A	443	320A
547	Female	3/25/34	433A	427	320A
----*	Female	1/8/35	433A	480	320A
----	Female	7/7/35	433A	537	433A
----	Male	7/19/36	433A	526	433A
----	Male	1/22/37	433A	587	433A
159**	Female	8/25/39	344A	See pedigree	
----		?	403B	----	----

*Calf shown in Figure 3.
**Calf lived, described in text.

TABLE II. Summary of the Results of Three Types of Matings

Results of mating a heterozygous bull to daughters of a heterozygous bull, as in pedigree B.

	Normal		Defective	
	Male	Female	Male	Female
Observed	19	11	1	2
	30		3	
Expected (one-eighth of progeny to be defective)	28.875		4.125	
d	+1.125		—1.125	

Results of mating a heterozygous bull to daughters and granddaughters of known heterozygous bulls, as in pedigree D.

	Normal		Defective	
	Male	Female	Male	Female
Observed	3	8	2	3
	11		5	
Expected (one-seventh of progeny to be defective)	13.7		2.3	
d	—2.7		+2.7	

Results of mnting a heterozygous bull to daughters, granddaughters and great-granddaughters of known heterozygous sires, as in pedigree E.

	Normal		Defective	
	Male	Female	Male	Female
Observed	3	5	1	0
	8		1	
Expected (seven forty-eighths of progeny to be defective)	7.7		1.3	
	+0.3		—0.3	

and Brandt[1] are compared with the cases observed in our Jerseys, differences become apparent. The defective calves exhibiting the Telemark type of achondroplasia have short, stumpy legs that will not support the weight of the body. The modifications of the skull are marked and follow a definite pattern. All the calves are very uniform in general phenotypic expression.

The defective calves observed in our Jersey herd are rather variable in this respect. All have the short broad head. Cleft palate and other associated deformities of the mouth are common, though not always present. Legs may be slightly reduced in length; but since the reduction is not marked, leg length cannot be used as a diagnostic character. The legs can bear the weight of the body in the milder cases. Although the more extreme types are lethal, the milder ones may live. The anomaly has its greatest effect on the development of the bones of the skull and maxilla, and little, if any effect on the length of the appendicular skeletal structures. Because of the phenotypical differences that have been pointed out (based primarily upon leg length) the authors believe that there are two recessive types of achondroplasia in cattle — the Telemark type and the one herein reported.

Punnett[9] reports results of crossing Dexter cows with a Telemark bull that was a proved carrier of the recessive Telemark type of achondroplasia. Though the data were somewhat fragmentary, the results clearly indicated that the two types of achondroplasia do not belong to an allelic series and that two independent loci are involved. If the type of achondroplasia herein reported could be crossed with the Dexter and the Telemark types, the genetic relationships of the three could be definitely established.

Summary

Inbreeding in Jersey cattle brought to light a sublethal type of achondroplasia that is rather variable in phenotypic expression. The greatest modification appears in the development of the bones of the skull and jaws. Defective calves may be identified by a short, broad head and a prominent forehead. Cleft palate may be observed in the more severe cases. The gene has little, if any, effect upon leg length. One female exhibiting a mild form of achondroplasia lived to 14 months of age until she was slaughtered.

This defect is inherited as a monofactorial autosomal recessive. It appears to differ from the Telemark type of recessive achondroplasia.

Literature Cited

1. Brandt, Graydon W. *Jour. of Hered.* 32(6):183-186. 1941.
2. Carmichael, J. *Nature* 131:878. 1933.
3. Crew, F. A. E. *Proc. Roy. Soc., B.* 95: 228-254. 1923.
4. Downs, W. G. *Anat. Rec.,* 37(4):365-372. 1928.
5. Eaton, O. N. *Jour. of Hered.* 28(9): 320-326. 1937.
6. Hutt, F. B. *Cornell Vet.* 24:1-25. 1934.
7. Mohr, O. L. *Zeitschr. f. indukt. Abstamm. u. Vererb.* 41:59-109. 1926.
8. Mohr, O. L. IV. *Congresso internazionale Di Patologia Comparate Roma. Estratto dal.* Volume I. 247-263. 1939.
9. Punnett, R. C. *Jour. Genet.* 32:65-72. 1936.
10. Wriedt, Chr. *Zeitschr. f. Tierzucht. u. Zuchtungsbiol.* 3:223-230. 1925.

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Eugenic Sterilizations Under State Laws

The Human Betterment Foundation of Pasadena, Calif., has issued its annual summary of legal sterilizations up to January 1942. In 1941 there were 2,209 sterilizations (652 in California). The total for 30 States, of which 28 now have active laws for sterilization, is 38,087 (15,780 males, 22,307 females). California leads with a total of 15,220; Virginia is second with 4,227. Other states with significant totals are: Kansas (2,583); Michigan (2,263); Minnesota (2,010); Oregon (1,542); Wisconsin (1,219); North Carolina (1,199) and Indiana (1,128). The complete table and related literature may be obtained without cost from the Foundation. (M.A.B.)

INHERITANCE OF MATURE PLANT CHARACTERS IN SORGHUM

Induced by Radiation

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THE discovery that mutations can be produced by irradiation has afforded opportunity for anyone interested in a particular plant species to create a number of abberrant races that may be used in analyzing the genetic constitution of that species. Additional mutated genes can be of material aid in chromosome mapping, and there remains the remote possibility that mutations favorable to plant growth and development might be produced. With these points in mind, different lots of dormant seed of sorghum (*Sorghum vulgare*, Pers.) have been subjected to X-radiation and the mutations observed for several years. Numerous mutants have been produced among which were several new and interesting mature plant characters which are described here and their inheritance reported.

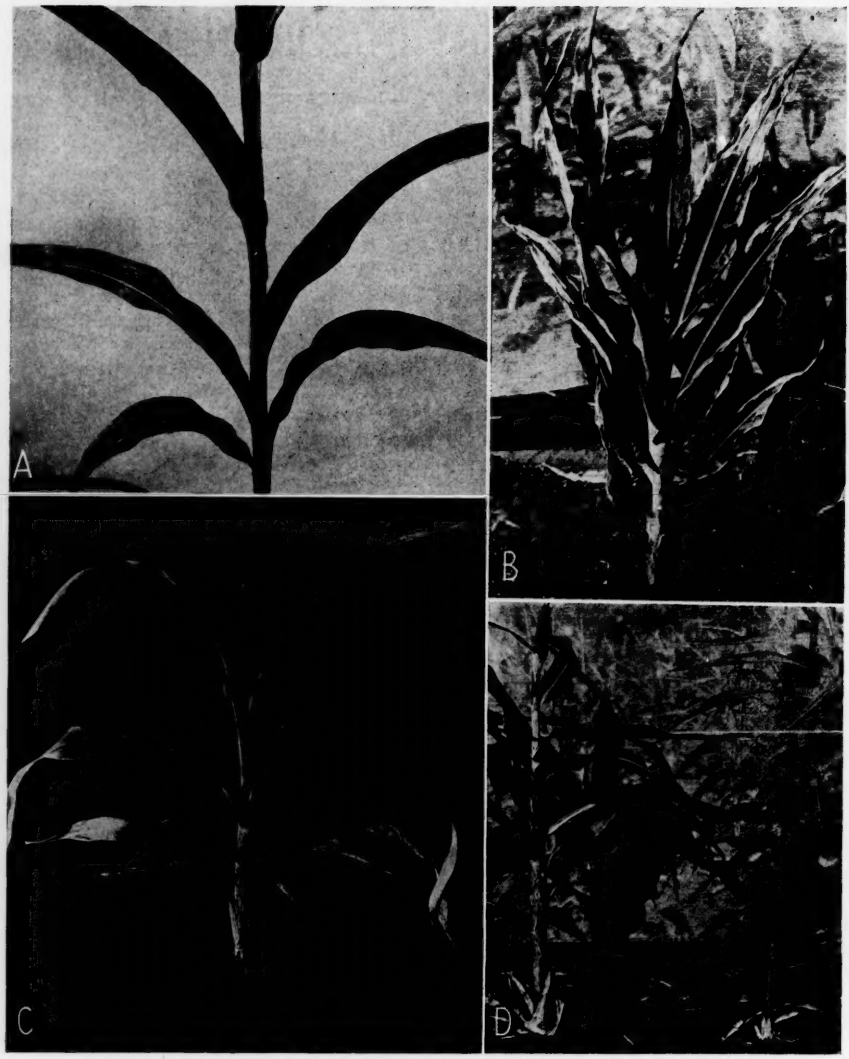
Mutations Produced by X-Radiation

During the progress of this work, approximately 2,000 progenies have been grown to maturity and 72 abnormalities recovered from them. The preponderance of these abnormalities are seedling characters of the chlorophyll deficient type. This paper is not concerned with these seedling characters, most of which are albinos, yellows, and virescents but these abnormalities will be reported as they are discovered to be linked with other genes and located on the chromosomes. Nine mature plant characters, two of which are discernible in the seedling stage, have been obtained and their appearance and inheritance are described in this paper.

Zebra Stripe

The zebra stripe (*zb*) character appeared in a stock homozygous for *virescent-2* (Karper and Conner[3]). In the field the zebra stripe character appears in the homozygous virescent stock in about 40 days at which time the plants have about eight leaves. In the F_2 generation of crosses involving this character, green plants homozygous for zebra stripe begin to show the character in about 30 days, the difference in time of appearance being due to the retarding effect of the virescent-2 gene. The expression of this character is not dependent upon the presence of the virescent-2 gene and it persists throughout the life of the plant after it has appeared. The character gets its name from the fact that there are transverse yellow or greenish-yellow stripes on the leaves caused by the dilution of chlorophyll in these areas (Figure 5*A*). This abnormality will usually appear on all leaves of a recessive plant but occasionally a plant is found with leaves almost entirely green, but with one or more unmistakable stripes. There is considerable variation in the amount of striping under different conditions. At Chillicothe, the leaves usually have from four to five stripes but as many as nine stripes have been observed at College Station. The number of stripes on the leaves is much less variable on the different leaves of the same plant. On mature leaves the streaks are usually two to three inches wide, separated by green areas of similar width. The zebra stripe gene in the homozygous condition reduces the vigor of plants somewhat. This reduction in vigor is apparent when a line homozygous for both virescent-2 and zebra stripe is compared with a line homozygous for virescent-2 alone, but is not so apparent in normal stocks.

Zebra stripe is a simple recessive to normal green. The counts in eight progenies as shown in Table I do not



RADIATION-INDUCED RECESSIVE CHARACTERS IN SORGHUM

Figure 5

A—A Zebra-stripe plant. Transverse greenish-yellow stripes two to three inches wide appear on all leaves of mature plants. *B*—A Fired-leaf plant with the margins of all leaves dead. The plants are stunted and ragged. *C*—Yellow leaf-tip plant seven weeks old. The yellow coloration develops progressively from the bottom to the top leaves and from the tip of the leaf toward the base. *D*—Mature Midget plants have from two to three times as many leaves as normal plants. The leaves are short and erect.

SEEDLING SEGREGATION

Figure 6

Normal and midget plants can easily be separated as seedlings. In a segregating population a close approximation to a 3:1 ratio is realized.

deviate significantly from a three to one ratio but in several individual rows there was a significant shortage of zebra stripe plants. Demerec[1] reports the inheritance of a zebra stripe in corn which this zebra stripe in sorghum resembles in all details. There is also a seedling zebra stripe in corn reported by Hayes[2] which is an entirely different character from the zebra stripe reported by Demerec. No counterpart of the seedling character of corn has yet been found in sorghum.

Midget

Plants homozygous for the gene that produces this abnormality called midget (*mi*) are extremely dwarf and at maturity rarely exceed 15 inches in height. The abnormality is recognizable in the seedling stage and can be readily separated from normal plants in seedling populations (Figure 6). By the time four leaves are present the shortening of the nodes is already evident, and this becomes more marked as growth proceeds. In addition to short stature, midget plants have more leaves than normal plants and, therefore, more nodes. From 20 to 30 leaves are usually present, but 40 leaves are not uncommon and the numerous short, straight, stiff leaves crowded on the stalk give the abnormal plants a distinctive appearance (Figure 5*D*). The peduncle is greatly shortened, although the usual dwarfing in sorghum is not accompanied by a corresponding shortening of the peduncle. This short peduncle of midget plants results in poor exsertion of the head from the boot and prevents seeds production in a large part of the head that does not clear the boot. Seed from midget plants is much smaller than normal seed, being about half of normal seed in weight. In many instances seed from midget plants germinates poorly but homozygous stock of this abnormality can be maintained. None of the mutant dwarf types reported as occurring in corn appears to resemble midget sorghum plants.

Midget is a simple recessive to the normal condition as shown by seedling counts that do not deviate significantly from a 3 : 1 ratio. In segregating populations at maturity, however, there is a marked shortage of recessive plants due to the inability of many of the homozygous recessives to survive.

Yellow Leaf Tip

This yellow leaf tip (*yl*) begins to show about a month after the plants

TABLE I. Inheritance of X-radiation induced characters.

Character	No. of Progenies	No. of plants		Percent Recessive	P
		Normal	Recessive		
Zebra	8	330	91	21.6	.1
Midget	11	1374	501	24.1	.30
Yellow leaf	4	336	58	17.3	.01
Fired leaf	4	497	122	19.7	.01
Mottled leaf	1	176	40	18.5	.02
Red leaf	1	171	34	16.6	.01
Freckled leaf	1	96	34	26.1	.70
Red leaf	1	72	22	23.4	.70
Dwarf	1	102	34	25.0	.99

AN INHERITED MICROMELIA IN THE DOMESTIC FOWL

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SEVERAL inherited skeletal abnormalities are known in the domestic fowl, but only in the case of sticky embryos[3] has any modification in calcification of the skeleton been observed. The mutation reported on in this paper was briefly described elsewhere.[2]

Material and Methods

All incubated eggs from the breeding flocks of the Division of Poultry Husbandry of the University of California at Davis that failed to hatch have been opened for examination during the past five years. The ratios for normal to abnormal embryos presented here include all chicks hatched and all embryos sufficiently well developed to classify.

Bones measured and analyzed were from embryos preserved in alcohol or from fresh specimens. The bones were freed from adhering tissue and measured. They were then extracted continuously in a Soxhlet apparatus for 24 to 48 hours with ethyl ether. The extracted bones were then placed in tared crucibles, dried at 100° C. for 12 hours, and weighed. The bones were ashed in a muffle furnace maintained at a temperature of 750° C. for one hour and again weighed.*

Description and Data

The skull of micromelic embryos is usually vaulted and the upper beak is curved and short (parrot beak); the lower beak is also short and typically deformed, while the legs are short and thick (Figure 7, Table I). Segregating families in 1938 to 1940 inclusive which gave both normal and abnormal progeny that survived past the 14th day of incubation produced 716 non-micromelic embryos and chicks and 47 micromelic embryos. This is a close approximation to a ratio of 15 normal to 1 abnormal (expected 715.9:47.7). If only families with two or more micromelic embryos are included, the numbers of progeny are 616 non-micromelic to 41 micromelic, or the same ratio as before. The ratios in individual families with two or more micromelic embryos vary from 5.75:1 (23:4) to 24.7:1 (74:3). Sex was determined for 40 of the 47 micromelic embryos, and of these 22 were males, 18 were females. The data, therefore, suggest that the micromelic embryos described above are double autosomal recessives rather than simple autosomal recessives as supposed on the basis of incomplete summaries.[2] It might be supposed that early embryo mortality would change the figures given materially. This is improbable, however, since 42 of the 47 micromelic embryos were alive at the 14th day of incubation and many of the 42 were alive on the 22nd day of incubation. Of the eggs allowed to hatch, as high as 89 per cent of fertile eggs have hatched from hens that, so far as known, carry no other lethal genes than those for micromelia.

Micromelic embryos were first found among the progeny of six females mated to a single male in 1938. The male and five of the females were out of an F_2 male (from an original cross of a Single Comb White Leghorn male mated to a Bantam female) mated to Single Comb White Leghorn females. The sixth was a half sister of some of these females but out of a Single Comb White Leghorn male and, therefore, herself a Single Comb White Leghorn. All the Leghorns belonged to the inbred strain of

*I am indebted to Dr. H. Goss and the Division of Animal Husbandry for the extractions of the bones and to Mr. D. C. Stoutamyer for preparing, measuring, and weighing the bones.

Single Comb White Leghorns that carried the amaxilla gene.[1] Six non-micromelic embryos and one micromelic were obtained from the one carrier Leghorn hen, but no tests are available for her progeny. An additional eleven hens have been found to be micromelia carriers; all of them descended from the original male mated to the hens out of the F_2 male or matings of these birds with other stock. No other male carriers have been found among the five tested. The male and seven of the seventeen micromelia carrier females are also heterozygous for the amaxilla gene.

It is entirely possible that some of the micromelia carrier hens so far tested are homozygous for one or the other of the two recessive genes presumably responsible for micromelia. If so, they should give seven normal to one micromelic embryos when mated to a male heterozygous for both pairs of genes. Some of the families fall within the range of about seven normal to one micromelic, as indicated above, but in view of the limited number of matings of carriers so far made a more detailed genetic analysis at this time appears unjustified.

Superficially the micromelic embryos look like the Cornish lethal embryo described by Landauer,[8] although they differ in the appearance of the beak. Crosses between stock carrying these lethals have given only normal progeny; hence they must be regarded as genetically distinct. The micromelic chicken embryos reported in this paper also resemble somewhat embryos that result from various nutritional deficiencies.[4,5,6,9,11] There are several significant differences, as follows: in the case of these nutritional deficiencies some of the embryos hatch; none of the micromelic embryos resulting from nutritional deficiencies figured in the various publications cited have the characteristically deformed lower beak shown in Figure 7; in the one case where bone ash was determined,[5] the abnormal embryos had a lower percentage of ash than normal embryos. Furthermore, if only nutritional or other environmental factors are involved in the production of the micromelic embryos described in this paper, such embryos should have been found in other families, all of which received identical rations and housing.

DOUBLE RECESSIVE MICROMELIA

Figure 7

Embryo of domestic fowl, showing micromelia, parrot beak, and deformed lower mandible.

The bones of the micromelic embryos were thicker than those of the normal embryos (Table I). Of the leg bones, the tibia was relatively most reduced in length (40.9 per cent of normal) and increased in diameter (178 per cent of normal). Of the wing bones, the humerus was reduced in length relatively more than the ulna or radius.

The bones of the micromelic embryos were reduced in weight. The dry weight of the more distal bones was in each case reduced relatively more in terms of the normal bones, the percentages being: femur, 85.9 per cent; tibia, 70.9 per cent; humerus, 80.3 per cent; radius-ulna, 61.3 per cent.

The amount of ash in the micromelic bones was approximately the same as that of the normal embryo bones. Since the dry weights of the micromelic bones were less than those of the normal bones,

the percentage of ash was distinctly higher for the micromelic than for the normal embryonic bones.

The data in Table I show that, at the time of hatching, the wing bones of chickens have a lower percentage of ash than the leg bones. In rats, on the other hand, Hammett[7] found that the percentage of ash was higher in the humerus than in the femur at 23 to 130 days of age with a much greater difference in the younger animals. Weakley and Dustman[12, 13] analyzed the bones of 193- to 288-day-old swine and five-week-old chicks and found only slight differences in per cent of ash in the femur and humerus. Their data indicate that the differences found in the long bones of gallinaceous embryos at hatching time tend to disappear by the time the birds are five weeks old, presumably because of a more rapid calcification of the wing bones following the hatching of the bird.

The type of micromelia described in this paper apparently reduces the amount of bone matrix without a corresponding reduction in the amount of ash, thus suggesting that the two—bone matrix formation and bone calcification—are to some extent genetically independent of each other. Landauer[10] has found that the greatest effect on the length of the long bones in embryos homozygous for the Cornish lethal gene is on the more distal bones. Thus, the tarso-metatarsus is reduced more in length than the tibia. In the micromelic embryos described in this paper the length of the tibia is reduced slightly more, though not significantly so, than the length of the tarso-metatarsus. The use of length of abnormal bones for comparison with normal bones as a measure of relative size would seem to be justified only where the relative diameter remains the same as in the normal. In the case of the micromelic embryos this is not the case; the weight of the leg bones was affected less than their shape since the increase in diameter of the micromelic bones partly offset their decrease in length.

Summary

Micromelic embryos with much shortened and thickened extremities and a typically deformed beak are described. Forty-seven micromelic embryos, comprising both males and females, segregated out of families producing one or more such embryos. There were 716 normal embryos, or a ratio of 15 normal to 1 abnormal, indicating that two autosomal genes are involved.

The percentage of ash was higher in the long bones of the micromelic embryos than in those of normal embryos, due to a reduction in bone matrix but not in ash.

The data presented show that in 22-day-old chicks the wing bones have a lower ash content than the leg bones.

Literature Cited

1. Asmundson, V. S. *Jour. Hered.* 27:401-404. 1936.
2. ————————. *Poultry Sci.* 18:399. 1939.
3. Byerly, T. C., and M. A. Jull. *Jour. Exptl. Zool.* 62:489-498. 1932.
4. Byerly, T. C., H. W. Titus, N. R. Ellis, and W. Landauer. *Proc. Soc. Exptl. Biol. Med.* 32:1542-1546. 1935.
5. Caskey, C. D., W. D. Gallup, and L. C. Norris. *Jour. Nutrition* 17:407-417. 1939.
6. Caskey, C. D., and L. C. Norris. *Proc. Soc. Exptl. Biol. Med.* 44:332-335. 1940.
7. Hammett, F. S. *Jour. Biol. Chem.* 64:409-428. 1925.
8. Landauer, W. *Jour. Genetics* 31:237-242. 1935.
9. ————————. *Anat. Rec.* 64:267-276. 1936.
10. ————————. *Storrs Bull. No. 288,* Conn. Agr. Expt. Sta., 1939.
11. Lyons, M., and M. M. Insko, Jr. *Kentucky Bull. No. 371,* pp. 62-75. 1937.
12. Weakley, C. E., Jr., and R. B. Dustman. *Agric. Res.* 58:711-716. 1939.
13. ————————. *W. Va. Bull. No. 294.* 1939.

TABLE I. Data on the long bones of normal and micromelic chicken embryos. The embryos were approximately 22 days old from eggs of similar size. Measurements are for 4 embryos per group, ash determinations for 2 chicks per group.

Description of Embryos	Femur	Tibia	Tarsometatarsus	Humerus	Ulna*	Radius*
			Average Maximum Length in Mm.			
Normal	19.9	27.4	20.2	13.3	12.2	10.7
Micromelia	9.1	11.2	8.8	6.2	6.5	5.4
			Average Diameter at Middle of Bone in Mm.			
Normal	1.61	1.69	2.20	1.15	0.92	0.61
Micromelia	2.79	3.01	3.20	1.46	1.23	0.73
			Average Dry Weight of Bone in Mgm.			
Normal	31.9	44.4		10.7	7.5	
Micromelia	27.4	31.5		6.6	4.8	
			Average Weight of Ash in Bones in Mgm.			
Normal	11.0	15.8		2.9	2.0	
Micromelia	11.2	13.5		2.9	2.0	
			Average Per Cent Ash in Bones.			
Normal	34.5	35.6		26.8	27.1	
Micromelia	40.9	42.9		43.7	41.0	

*The analytical data are for the combined ulna and radius.

MATING CUSTOMS IN NORTH CAROLINA*

1750-1900

FLORENCE C. DUDLEY AND WILLIAM ALLAN

THE distribution of pathological recessive genes and the appearance of resultant recessive traits depend, among other things, on the mating habits of a population. The marriage customs of our people at various times and in different parts of the country are thus important in formulating eugenic programs. In order to secure more definite information on this subject we have studied two samples of the populatiton in North Carolina: (1) the Hopewell Church congregation in Mecklenburg County, and (2) the population of Watauga County beyond the Blue Ridge, in the Smokey Mountain region. These two populations differ only in the length of time they have inhabited their respective localities. Both are composed of the descendants of immigrants from the British Isles and Germany.

Mecklenburg Congregation

The first land grant in Mecklenburg was dated 1749 and during the next forty or fifty years settlers gradually filled up the county and took up all the land.[2] These early settlers in Mecklenburg were predominantly Scotch Irish Presbyterians. By the time of the Revolution the population was grouped around the seven original Presbyterian churches of the county. Social life revolved around these churches.

Hopewell church, ten miles east of Charlotte, one of the original seven, was organized in 1762 and to this day has continued to be a vigorous organization. At first its territory extended ten to fifteen miles in every direction and the worshippers would spend the day riding on horse-back or driving wagons to church. Buggies appeared nearly a century later. As the population increased, other churches were established in the periphery of Hopewell's territory so that the size of the congregation has remained about the same through the years, from 250 to 300, representing about 80 families. Until 1900, life in Mecklenburg remained strictly rural. The county seat, Charlotte, had less than 1,000 white inhabitants after the Civil War. There was no church in town before 1825. Not only the churches, but schools, taverns, doctors, and many of the stores were located in the country.

The history of Hopewell church gives a good account of the early families down to the present time. We have examined these records down to 1900 with the help of a great-grandson of the man in whose house the church was started, to find out how often the boys and girls of that congregation married inside or outside the congregation. Of 446 marriages recorded, 339 or 76% of all the marriages by members of that congregation, were to members of the same congregation. This means that any recessive traits brought into that group before 1900 had three chances out of four of remaining confined and concentrated within that group. The effect of selective mating within a church congregation on the frequency of cousin marriages will be dealt with in a subsequent communication.

Three Forks Baptist Congregation

After the land of the Piedmont region of North Carolina had been occupied, new settlers, along with the children and grandchildren of the inhabitants of the middle counties, began to migrate to the Western Waters, taking up the land beyond the Blue Ridge. This movement started soon after the Revolution. The Three Forks Baptist Church, established

*From the Department of Medical Genetics, The Bowman Gray School of Medicine, Winston-Salem, N. C.

in 1790, was the first in Watauga county.[3] In this country of high mountains and narrow valleys, topography was the chief factor in causing isolation of the groups living up and down the creeks. The church tended to counteract this effect by assembling the people of the isolated communities for worship at a central location on Sunday. The older inhabitants of Watauga county give us the impression that young people living on the same creek were much more apt to marry than the members of the same congregation. We have no figures for Watauga based on either church membership or neighborhood residence, but the cousin marriage rate for this county, when worked out from the pedigrees, will prove to be remarkably high, although partly for a different reason. Watauga was settled between 1785 and 1820 by 100 to 150 pioneer men, probably no more than 50 of whom came with their wives. Since the end of that period almost no new settlers have entered the county, and today some 90 per cent of the inhabitants of this territory are the direct descendents of the small original group of pioneers. The Piedmont Carolina counties, then, from 1750 to 1900 consisted of a large number of small isolated communities grouped around the country churches. The mountain counties, starting a generation or two later have been made up entirely of similar small isolates, whose areas have been limited chiefly by the topography.

In attempting to get some idea of the size of these isolates we have asked many of the older men and women how many young people of the same age and opposite sex they knew well enough to pick a husband or wife from. We would ask a man, "How many girls did you call on, or take buggy riding in your courting days?" The answer ranged around a dozen or so, with the maximum set at two or three dozen.

In the western half of North Carolina from its settlement to the coming of good roads, automobiles, consolidated schools and the rise of towns, mating was highly selective and confined very largely to other members of small church or neighborhood groups. The idea of random mating among hundreds or thousands of people, as used in the genetic literature, when applied to uncommon pathological traits, turns out to be a fantasy of about the order of Alice in Wonderland in the area reported on here.

Literature Cited

1. Arthur, J. P. History of Watauga County. Everett Waddy Co., Richmond, Va. P. 71, 1915.

2. Summerville, C. W., Ph.D., D.D. The History of Hopewell Presbyterian Church. Observer Printing Office, Charlotte, N. C. 1939.

3. Tompkins, P. A. History of Mecklenburg County. Observer Printing House. Vol. 1, 1903.

THE SIZE OF MATING GROUPS

DUDLEY and Allan's study of the size of mating-isolates in North Carolina offers an important, and apparently completely neglected, lead to a new approach to preventive eugenics. Very little is known regarding the size of mating-isolates in other parts of the country ,and until such information is available, it is hardly possible to assess the conditions pictured for North Carolina as being average, modal, or extreme. Until data exist regarding mating-customs in a great many different kinds of social structures, the authors' relegation of genetical concepts to "Alice in Wonderland" may be a trifle premature. This criticism smacks a bit of the straw-man, because geneticists never have made any blanket-assumptions that all mating-isolates are made up of hundreds of thousands of people. Obviously they very often could not be.

A point which Dudley and Allan fail to make is that within the past few years a good deal of work* has been done by Fisher, Wright, Haldane, and other bio-

*The concept was considered in its mathematical implications by Wright, at a Symposium arranged by the Milbank Foundation (*New Health Frontiers,* pp. 20-22, Milbank Memorial Fund, New York, 1937), but this seems not to have been much noticed by eugenists.

metricians to explore the mathematical consequences of the now abundantly established pattern of mendelizing chromosomal heredity on the evolution of genic systems.

A basic concept in these calculations has been that every time a germ-cell is formed, one member of each chromosome-pair is discarded. This simple fact ramifies into some very complicated calculations when linkage and other complications set in, before one gets very far into population genetics. But some of the conclusions are quite simple and clear-cut.

One basic principle of these computations is that the survival as a result of random mating of any given mutation is tremendously influenced by the size of the mating-isolate. In a large interbreeding population, even a favorable mutation has a very slim chance of surviving the 50-50 chance it will be thrown away in any given mating. The only situation in which new mutations, and rare pathological genes new or old, are likely to survive this random-discard mechanism is *in small and isolated* mating-groups. When the geneticist's random-mating in big populations takes place, it is virtually impossible for the rare recessive gene to *survive*, let alone multiply.

Surely here is a principle that has the utmost eugenic significance, and one which might be more readily attacked than a good many eugenic problems. There is no doubt whatever that the situation reported by Dudley and Allan is the kind of situation the geneticist would set up to fix and perpetuate in a population recessive genes, whether old or newly mutated, good or bad. Allan has elsewhere reported, that the population he is studying does carry a number of pathological recessives. It is therefore of great significance that the pattern of closely interrelated small mating-islotes is the ideal way to assure that such recessive genes will produce the maximum misfortune.

The question of whether it would be possible to eliminate such nests of pathological genes by dilution through increasing the size of the mating-groups would be one for the biometricians to decide. Unquestionably with very rare genes this could be done. It would be quite simple if it were possible to identify the carriers of deleterious recessives, but even without that there would seem to be a good chance that merely through increasing the size of mating-groups, much could be done to reduce the percentage of defect, and even *to reduce the frequency* of morbid genes. Before one could speak authoritatively with regard to that the possibilities must be explored by the gene-frequency experts.

Following this line of thought brings one to some rather challenging speculations. Is it possible that the huge state universities take on a new potential significance as recessive gene-diluters? What of the geographical and familial relationships of the graduates of such colleges who marry? Surely the picture must be very different from what Dudley and Allan give us. It is very likely that the buggy-riding degree of closeness of acquaintanceship will not go so much higher than the three-dozen maximum reported by these authors. Not even the most active Lothario is going to take a hundred thousand girls for moonlight rides. That is mercifully not necessary to attain a state of random mating as far as a given recessive gene is concerned. By the same reasoning it is not necessary to have 100,000 boys and girls in college to have a random sample of a population of that size. Thus it might turn out on mathematical analysis that the founding of the Land-Grant Colleges in the United States was a eugenic expedient of the utmost importance.—R. C.

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COMMENT BY DR. SEWALL WRIGHT

I WAS much interested in the note by Dudley and Allan which you sent me and your comment. Their final sentence is, as you note, very much of a setting up of a straw man, at least as far as I am concerned, and certainly this holds for many other geneticists.

The questions of the chance of fixation

of a new mutation and the persistence of one that has become established at some appreciable frequency in a population are somewhat different. Following are the probabilities of fixation of a recessive gene in various population sizes, considering three cases, selective advantage of *aa* of .01, a selective disadvantage of .01, and neutrality.

RECESSIVE GENE

Size of Population	Advantage of aa = .01	Neutral	Disadvantage of aa = .01
10	.05	.05	.05
50	.013	.010	.007
200	.0057	.0025	.0003
800	.0027	.0006	.0000

A semidominant favorable gene rapidly approaches a constant chance of fixation (twice selective advantage of heterozygote) but even in this case the chance is better in a small population. Of course an unfavorable gene has an enormously better chance of fixation $(1/2n)$ in a small population than in a large one.

All of this ignores recurrence of mutation. An unfavorable recessive that keeps coming back by recurrent mutation reaches a certain equilibrium frequency $q = \sqrt{v/s}$ when v is the rate of mutation and s the selective disadvantage. This may be moderately high; e. g. if $v = .000.01$ and $s = .01$, then $q = \sqrt{.001} = .032$. This is of course merely gene frequency, actual recessive individuals have a frequency of only $q^2 = .001$. This is in a large population. In a small one this gene will usually be absent, but if present at all it will come to light much more often. If highly injurious—lethal or nearly so—it will be eliminated to a greater extent in a group of small populations than in a random-breeding large one. Thus Dobzhansky, Hovanitz, and I (*Genetics* 27:363-394 1942) have shown that lethals are not as common in *Drosophila pseudoobscura* as they would be if breeding were at random. We concluded that in the continuous population on Mt. San Jacinto, choice of mates was restricted to some 50 individuals or less.

Genes that are only slightly injurious could readily be fixed in small populations in which case they would not be subject to evolution at all *within* that population. But if there is intergroup competition—some populations increasing more rapidly than others and tending to displace them directly or by grading up (in spite of a high degree of isolation)—selection would be effective in a way that it would be impossible in a large population in which there can be no selection for genetic system as wholes.

The main effects of random mating—via the State Universities or otherwise—on an array of isolated communities would be to *decrease* the appearance of unfavorable recessive types and to *increase* the gene frequencies. I suspect that in the long run—evolution of man from ape—a certain degree of fineness of subdivision of the population is favorable. In the short run — which may mean a good many centuries—random mating would mean a greatly improved population. After a long period—when recurrent mutation has had time to bring the frequencies of unfavorable genes to some approach to their equilibrium points—the incidence of defects will begin to rise. At this stage the splitting up into small isolates would have effects horrible to contemplate.

Guinea pigs, rats, mice, etc., have probably always been inbred a great deal by fanciers. They stand up fairly well under brother-sister mating in consequence. Chickens have not been inbred much and consequently have so many injurious traits at moderately high gene frequencies that they go to pieces promptly under brother-sister mating (with rare exceptions). An extreme case is that of corn (usually cross bred) and wheat (usually self fertilized). The former rarely stands selfing for more than a few generations, but a mixed population produced from lines that have persisted at all under selfing is extraordinarily vigorous. — SEWALL WRIGHT, *University of Chicago.*

EDUCATION FOR FAMILY LIFE

A Review

SUGGESTING as it does, a primary concern with youth, the title of this outstanding volume* is somewhat misleading, for it is the most thoroughgoing and comprehensive treatment of family life education that has appeared so far. It extends from nursery education through adulthood, through all phases of education, social service, law, medicine, and religion that touch upon family life in local community, state, and nation. The discussion is enriched by descriptions of programs and techniques gathered from all over the United States, and deepened by philosophical searchings for underlying meanings and purposes. In spite of the great condensation of fact and thought in its 238 pages, the volume is easily read. The reviewer believes that *Youth, Family and Education* is the most valuable single reference in the field.

Current practices and needs are given significance by a background discussion of changing social problems. Family discord and disorganization and the declining birth rate in the upper cultural and economic levels are seen not only as social evils in themselves but as problems which cut into the very foundations of the future. As certain former functions of the family are taken over by other agencies, those which remain gain in importance. These are: "(1) the physical reproduction of the race, (2) the irreducible minimum of physical care and training of the young child, (3) provision of the fundamental sources of mental health and happiness for the great majority of persons—that is, a sense of personal security, enduring affection, and healthful sex life, and when the home fails in any of these functions, numerous individual and family disasters follow at severe cost both to family members and to community." (p. 28.)

As long as we wish to preserve the democratic way of life (and we are waging mighty battles in both oceans for that very purpose) we cannot *force* people to maintain wholesome homes and rear an adequate number of children. We can oly inspire them by making clearer the importance of family living ,and by improving the number and quality of satisfactions it provides, through educational media. "Through intelligent reorganization of housework, through more adequate measures of social security, and through education and guidance for family living begun in the earliest years and carried on through maturity, family life and child rearing can be made a genuinely satisfying experience which will be looked upon as a privilege rather than a burdensome duty. The most important mission of family life education is this mission which some have called eugenic." (p. 238.)

From different approaches, educators and social workers have come to the same conclusions as to the importance of education for the family. And it is proving itself an effective instrument both for the actual improvement of family life and for increasing the desire of modern young people to have happy families of their own. Dr. Folsom reports several studies which indicate an increasing interest in and desire for family life among the young people of today.

Fortunately, during the last 30 years scientific findings regarding human growth and relationships have increased enormously, and are available for the questionings of youth. Education can improve material skills, but it may also improve skills in living such as developing a satisfying love life, dealing with a sick or irritable child, or a jealous spouse, or with life adjustments after forty.

In spite of this larger fund of knowl-

*FOLSOM, JOSEPH K. *Youth, Family and Education.* American Council on Education, Washington, D. C. 1941.

edge, it is emphasized that education for family life does not consist primarily of handing down wisdom to the young. It is rather a joint searching of old and young together for the deeper meanings and values to be found in the family.

The volume is enriched by Winfred Bain's article on nursery education and Ellen Miller's discussion of family life education at the elementary school level. Miss Bain emphasizes parent education as a most important function of nursery schools, holding that the care of little children in the home should be considered a skilled professional activity. Ellen Miller's discussion of family life education in the elementary school is the most complete and authoritative in the field. She points out that the child's family experience should not be used merely as a point of departure for exploring other fields, but as study material of first importance in helping children grasp the deeper meaning of important realities such as mother, father, home. Whether or not this is accomplished is determined by the interest and insight of the teacher rather than the type of curricular organization.

The amount and variety of family life education in the high schools is encouraging. It is included at various points in home economics, the social sciences, biology, health education, mental hygiene and some guidance programs. When available, nursery schools have proven extremely valuable as practice and observation centers for both boys and girls. In some systems there is excellent "practice-house" experience where boys and girls work together in purchasing, planning, cooking, etc., in addition to classroom study of budgeting and home management. Even though excellent work is carried on in many cities it is estimated that only ten per cent of the high school students are reached by any family-life instruction.

There has been a truly remarkable development of family life education in colleges during the past fifteen years, not only in pointing up what was already there but also in new programs and courses. Folsom found family life courses in sixty-four per cent of the women's colleges and twenty-seven per cent of the men's. Although sex instruction is the main interest of most students studying family life, there is also a very real need for instruction in the economic, parental, emotional adjustment and community relations phases of family living.

Obviously no pre-parental education can fully prepare individuals for all the problems they will meet later in rearing families. There is and will continue to be great need for adult education not only for young married couples but for those all along the road of life's manifold changes and ever new adjustments. The various phases of adult education both in group work and in various types of counseling are sympathetically dealt with in two chapters on these subjects. The mental hygiene value of adult groups where parents find many others facing the same problems is very real.

There are a number of community, state and national organizations concerned with the problems of family life education programs. Dr. Folsom contends, however, that while some overhead functions are adequately performed by the diverse organizations now existing, others need further distribution or integration. These are carefully analyzed in a chapter on "The Distributive Functions in a National Program."

But throughout the whole area of family life education, be it in formal courses and counsel, or casual education through personal contacts and recreation, the process must be the democratic one of liberating the forces in every individual that seek ever truer values for human life. As Folsom puts it "we are now rightly placing less emphasis upon 'ideal' family life, fixed rules, and the results of statistical research. We are emphasizing growth and development, the interaction of persons, the total family-community situation, the influence on our culture upon our attitudes, and the need for facing openly the conflicts between values." (p. 232.)

KATHARINE WHITESIDE TAYLOR
Consultant in Family Life Education
Seattle Public Schools

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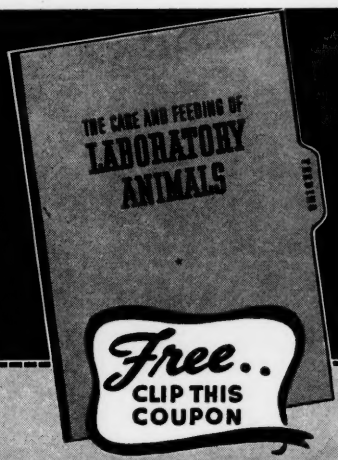

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